ductive process is involved. In all cases, homogenates of diabetic liver showed a marked increase in CO_2 fixation.

Incorporation of $^{14}CO_2$ into oxaloacetate by whole liver homogenates was determined by incubation in the presence of pyruvate and ATP. An aliquot of the supernatant fluid was counted in anthracene-packed cuvettes in a Packard Scintillation Counter before and after treatment with aniline hydrochloride[3]. The difference in radioactivity was presumed to be due to ^{14}C incorporation into oxaloacetate. In homogenates of diabetic livers 7750 ± 780 counts/min were incorporated as compared with 1050 ± 110 counts/min in homogenates of normal liver.

Phosphopyruvate carboxylase (EC 4.1.1.32) was assayed in livers of normal and alloxan-diabetic rats by the method of UTTER AND KURAHASHI[4]. Livers were homogenized in 0.154 M KCl and centrifuged at 105 000 × g for 60 min 0.1 ml of the supernatant fluid equivalent to 0.033 g of fresh liver was incubated with 5 μC of [^{14}C]bicarbonate, 20 μmoles of oxaloacetate; 2 μmoles of $MnCl_2$ and with or without ITP (2 μmoles). At the end of a 10-min incubation solutions were deproteinized with trichloroacetic acid and gassed with CO_2. An aliquot of the filtrate was counted in a Packard Scintillation Counter. Phosphopyruvate carboxylase activity was found to be increased 300 ± 25% when expressed per g of wet liver in alloxan-diabetic animals as compared to normal.

These results indicate that in the liver of alloxan-diabetic rats there is an increase in CO_2 fixation and phosphopyruvate carboxylase activity. It is suggested that the increase in gluconeogenesis in the diabetic may be in part due to an increased conversion of lactate and pyruvate to phosphoenolpyruvate.

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The iodide pool of the thyroid studied by means of the isotopic equilibrium method and double labelling with ^{125}I and ^{131}I

The amount of iodide present in the thyroid is very low. It is renewed in two ways: by uptake from the plasmatic iodide and by deiodination of the iodotyrosines (recycling). In the present work, the amount of iodide present in the rat thyroid has been measured by the isotopic equilibrium method[1–3] without any pharmacological intervention. By combining this method with double labelling it has been possible to study the kinetics of iodide turnover and to estimate the importance of recycling.

Rats fed on an iodine-free diet were given a constant daily dose of iodine in their drinking water in the form of KI. One group received 5 μg of iodide per day, a second group received 50 μg. In animals previously adapted in this way, isotopic equilibrium was reached by a 50-day treatment with the same drinking water labelled with ^{125}I at a constant specific radioactivity (in μC/μg iodide) except for the physical decay of the radioisotope. This method is preferable to that in which the stable iodine and the tracer are given separately, as it assures that the precursor ingested is taken at a constant specific radioactivity. Thus when isotopic equilibrium is obtained, each pool of the gland is uniformly labelled at the specific radioactivity of the drinking water. The iodine pools can therefore be calculated quantitatively from the ratio of their radioactivity to the specific radioactivity of the drinking water.

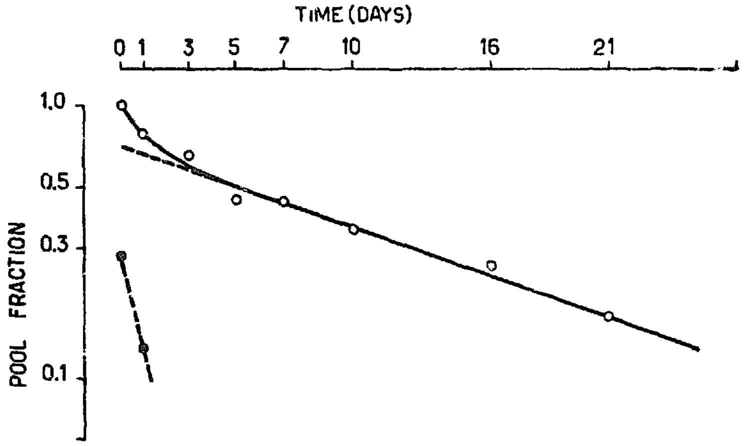

Fig. 1. Thyroidal iodide turnover. Rats fed 5 μg iodide per day. O—O, F_2 = 0.71, $(T_{½})_2$ = 10 days, α_2 = 0.069 days^{-1}. ●---●, F_1 = 0.29, $(T_{½})_1$ = 1.0 days, α_1 = 0.69 days^{-1}.

Iodide turnover was studied in the following way. While maintaining the first isotopic equilibrium with ^{125}I, the establishment of a second isotopic equilibrium was begun with the isotope ^{131}I. The rats were killed in groups of 3 or 4 over a period of 1–21 days after the beginning of ^{131}I labelling. After each time interval *t*, the ratio of the ^{131}I radioactivity to the specific radioactivity (as regards ^{131}I) of the drinking water is a measure of the amount of the iodine renewed by the end of *t* days.

The separation of the iodide pool was carried out on homogenized glands by paper electrophoresis with 0.2 M ammonium carbonate buffer (pH 9.4) (ref. 4) over a period of 1 h at 150 V and 10 mA. The separated iodide and total organic iodine spots were detected by autoradiography (Kodirex no screen) and cut out. Their ^{131}I and ^{125}I radioactivities were measured by means of a well-type scintillator.

The total quantity of iodine contained in each gland was calculated from ^{125}I measurements before electrophoresis. The percentage of iodide can be calculated from the measurement of ^{125}I on the electrophoregrams. For the rats fed 5 μg of iodine per day (22 animals), 0.30 ± 0.03 (S.D.) per cent of the total iodine of the thyroid (10.5 ± 2.1 μg iodine) is in the form of iodide, *i.e.* 0.0316 ± 0.008 μg. For the rats fed 50 μg of iodine per day (26 animals), 0.45 ± 0.01 per cent of the total iodine of the gland (15.9 ± 2.3 μg) is in the form of iodide, *i.e.* 0.0709 ± 0.0234 μg. The sodium space (^{24}Na) of the thyroid of 11 animals was measured; from this it was concluded that the maximum possible contamination by extracellular iodide was negligible.

The renewed fraction $F(t)$ of the iodide pool for time t is represented by the ratio of the amount of iodide renewed at time t to the value of the iodide pool. If the function $1-F(t)$ is plotted on a semi-logarithmic scale, the results are as given in Figs. 1 and 2. A parallel study of the organic iodine pool of the gland (results to be published) showed that the lowest iodide turnover rate, α_2, is equivalent to that of the organic pool. The pool fraction F_2 obtained by extrapolation at zero time, thus represents the part played by deiodination of the iodotyrosines in the turnover of the iodide pool. In the rats fed 5 μg of iodine per day, 71 % of the iodide concerned in the biosynthesis of the thyroidal hormones is derived from recycling ($F_1 = 0.29$; $F_2 = 0.71$). For rats fed 50 μg of iodine per day, only 47 % is derived from the deiodination of the iodotyrosines ($F_1 = 0.53$; $F_2 = 0.47$).

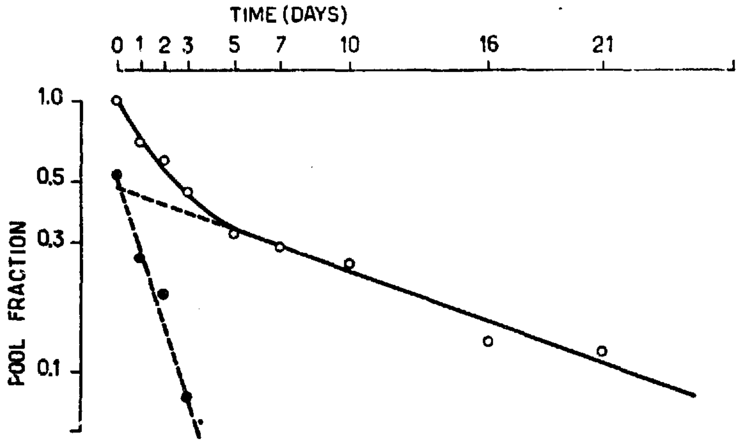

Fig. 2. Thyroidal iodide turnover. Rats fed 50 μg iodide per day. O—O, $F_2 = 0.47$, $(T½)_2 = 9.8$ days, $\alpha_2 = 0.071$ days^{-1}. ●---●, $F_1 = 0.53$, $(T½)_1 = 1.0$ days, $\alpha_1 = 0.69$ days^{-1}.

These results support the hypothesis of a single iodide pool in which the turnover is determined by two fluxes (recycling and uptake) whose relative participation has been estimated. The quantitative determination of the amount of the thyroidal iodide is approximately in agreement with its estimation by HALMI AND PITT-RIVERS[5] who have assumed that iodide represents 0.26 % of the total amount of iodine of the thyroid, the higher values observed here being explained by the fact that a true state of isotopic equilibrium is reached in the present work.

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